CLINICAL, RADIOLOGICAL AND ELECTRO PHYSIOLOGICAL STUDY IN PATIENTS WITH COMPRESSIVE AND NON COMPRESSIVE MYELOPATHY

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CERTIFICATE

This is to certify that this dissertation entitled **"CLINICAL, RADIOLOGICAL AND ELECTRO PHYSIOLOGICAL STUDY IN PATIENTS WITH COMPRESSIVE AND NON COMPRESSIVE MYELOPATHY"** submitted by Dr K.Sankara Subramanian appearing for D.M. (NEUROLOGY) Degree examination in August 2012, is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr.M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that this dissertation titled **"CLINICAL, RADIOLOGICAL AND ELECTRO PHYSIOLOGICAL STUDY IN PATIENTS WITH COMPRESSIVE AND NON COMPRESSIVE MYELOPATHY"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of **Prof. Dr. K.Bhanu, M.D., D.M., Additional Professor of Neurology, and Prof. Dr. R.M. Bhoopathy, M.D., D.M., Professor of Neurology**, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the university requirements for the award of the degree of D.M.Neurology.

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CONTENTS

ABSTRACT

CLINICAL, RADIOLOGICAL AND ELECTRO PHYSIOLOGICAL STUDY IN PATIENTS WITH COMPRESSIVE AND NON COMPRESSIVE MYELOPATHY

BACKGROUND:

The etiological spectrum of spinal cord disease or myelopathy is diverse. Broadly they are classified into compressive and non compressive myelopathy. The clinical presentation of these spinal cord diseases are diverse . With the advent of Magnetic Resonance Imaging (MRI) diagnosis of myelopathy has become easier. But there are certain diseases where at times the MRI can be normal and Somatosensory Evoked Potentials (SSEP) help in diagnosing the involvement of spinal cord

AIMS AND OBJECTIVES:

To study and analyse the clinical and radiological features of compressive and non compressive myelopathy .To determine the utility of SSEPs in compressive and non compressive myelopathy

MATERIALS AND METHODS:

Fifty two patients with features of myelopathy were included (compressive myelopathy-26 and noncompressive myelopathy- 26) in the study. Relevant blood investigations , Cerebrospinal fluid analysis , MRI and SSEPs were done. Twenty age and sex matched

controls were included in the study and SSEPs were done for them. Patients with coexisting neuropathy,radiculopathy, plexus lesion , brain stem and cerebral cortical lesions were excluded.

RESULTS

Cervical spondylosis was the most common etiology of compressive myelopathy followed by Tuberculosis of spine. Acute transverse myelitis was the commonest cause among the non compressive myelopathies. Median SSEPs showed absent N13 potential in cervical spondylotic myelopathy with cord compression at C5,C5 segment. In acute transverse myelitis the thoracic cord was the commonest site of involvement and tibial SSEPs showed prolonged central conduction time from N20 (T12 level) to N28(C5 segment). SSEPs were helpful in identifying the cord involvement in subacute combined degeneration (SACD) and HIV myelopathy patients were MRI was normal.

CONCLUSION

In patients with cervical spondylotic myelopathy(CSM) who had intact posterior column sensations median SSEP´ s were abnormal indicating subclinical involvement. Absent or prolonged latencies of N13 potential in median SSEP correlated with absent or reduced tendon reflexes of C5 and C6 segment. In patients with subacute combined degeneration of the cord and HIV myelopathy having normal MRI, posterior column involvement is indicated by abnormal SSEPs.

INTRODUCTION

Spinal cord disease or myelopathy contributes to a significant proportion of cases in neurology practise. Patients with myelopathy present with varied clinical manifestations depending on the level of lesion , the involvement of ascending and descending tracts of white matter and grey matter.

Motor dysfunction with paraplegia or quadriplegia can occur. Sensory impairment below the level of lesion is the usual presentation. It may be modality specific depending on the involvement of spinothalamic or posterior columns. Autonomic involvement can cause bladder, bowel, sexual dysfunction and also abnormalities in sweating , piloerection and vasomotor tone .

Myelopathies can be classified into compressive and non compressive myelopathy based on the etiological factors. In compressive myelopathy patients usually present with vertebral pain , radicular pain or a funicular pain with a definite sensory level below which either the posterior column or lateral spinothalamic tract sensations or both are lost. Compression of the cord can arise from the cord(intramedullary) or outside the cord(extramedullary). The commonest cause of compressive myelopathy include the degenerative disease of the spine I.e)the

spondylotic myelopathy. In developing countries where tuberculosis is still common , involvement of the vertebra results in pott s spine and compression of the cord. Other causes include vertebral metastasis with cord compression, traumatic myelopathy, arachnoididtis and primary intradural nerve sheath tumors

Non compressive myelopathy encompasses various diseases such as acute transverse myelitis, primary demyelinating disorder such as multiple sclerosis and neuromyelitis optica ,subacute combined degeneration of the cord , HIV myelopathy , radiation and toxin induced myelopathy and degenerative diseases of tbe cord. Except for transverse myelitis other non compressive myelopathy do not present with a sensory level. Extensive blood and CSF examinations may not reveal the etiology. Even magnetic resonance imaging of the spine will be normal. So electrophysiological studies such as somatosensory evoked potentials(SSEP) may help to localise the site of lesion in the spinal cord

Myelopathies result in motor disability thereby restricting patients mobility and lead to a poor quality of life. Hence prompt and timely intervention is important to prevent major disabilities.

AIM AND OBJECTIVES OF THE STUDY

- To study and analyse the clinical and radiological features of compressive and non compressive myelopathy
- To determine the utility of somatosensory evoked potentials in compressive and non compressive myelopathy

REVIEW OF LITERATURE

Spinal cord anatomy:

It extends from the foramen magnum above and terminates in the lower border of L1 vertebra. The average length of the spinal cord in an adult male is 45 cm and in the adult female it is 42-43 cm.[1,2] The corresponding length of vertebral column is 70 cm. The spinal cord is enlarged from C5-T1 and L3-S2 forming the cervical and lumbar enlargements from which arise nerve supply to the extremities. Below the lumbar enlargement the spinal cord forms the conus medullaris. After L1 vertebra the pia matter of the spinal cord tapers to form filum terminale. It is enclosed within the spinal canal formed anteriorly by the vertebral body , laterally by the pedicle and laminae and posteriorly by the spinous process. The spinal cord is enclosed in meninges and protected by the posterior longtitudinal ligament and ligamentum flavum. The intervertebral disc consists of inner nucleus pulposus and outer annulus fibrosis.

The spinal cord is composed of an inner core of gray matter, which is surrounded by an outer covering of white matter. The gray matter is seen as an H-shaped structure and divided into anterior and posterior gray columns or horns. The white matter is divided into anterior, lateral,

and posterior white columns or funiculi. The posterior column carries fibers for proprioceptive sensation. The lateral column consists of lateral spinothalamic, corticospinal , rubrospinal and reticulospinal tract.

Ascending tract of spinal cord

1.Lateral and ventral spinothalamic tract

2. posterior column- fasciculus gracilis and fasciculus cuneatus

3. dorsal and ventral spinocerebellar tracts

Descending tract of spinal cord

1.Corticospinal tract

2.corticorubrospinal tract

3.lateral reticulospinal tract

4.medial reticulospinal tract

5. vestibulospinal tract

Blood supply of the spinal cord is mainly through one anterior spinal artery and two posterior spinal arteries.[3] They arise from the vertebral arteries. They anastomose with radicular arteries which enter

the intervertebral foramen along with nerve roots forming a arterial network on the surface of spinal cord

The clinical manifestation of spinal cord disease is classified based on the tract involved.

1. Complete transection of the spinal cord (transverse myelopathy)
2. Brown –Sequard syndrome (hemisection of spinal cord)
3. Central cord syndrome
4. Posterolateral column syndrome
5. A combination of pyramidal tract and anterior horn cell disease

Complete transection of the spinal cord

The clinical presentation of transverse myelopathy is florid since there is involvement of all ascending and descending tracts at the level of involvement of cord. All modalities of sensation including soft touch, position sense, vibration , temperature and pain are impaired below the level of the lesion.[4] Band like sensation or segmental paresthesia usually occur at the level of lesion.[5] This clinical syndrome is well described in traumatic spinal cord injuries which is the most common cause .Similar presentation also occurs in inflammation or demyelinating lesions,

hemorrhage and infarct of the cord and radiation myelopathy which can evolve subacutely.

Stage of spinal shock

Paraplegia or quadriplegia occur due to the involvement of the corticospinal tract below the level of the lesion. The muscles are flaccid and all spinal segmental reflex are absent below the level of lesion. Paralysis of bladder and bowel with gastric atony, occurs Other autonomic functions such as vasomotor tone, sweating and piloerection are also absent below the level of the lesion. As a result of this the skin becomes dry and swelling of legs develops. The sphincters of bladder and bowel are contracted because they are disconnected from the higher inhibitory centres and the detrusor and smooth muscle of rectum are atonic. So there is accumulation of urine until the intravesicular pressure overcomes the sphincter to cause overflow incontinence. The reflex activity starts to appear after 1-6 weeks. Bulbocavernosus reflex is the first to reappear. The physiologic basis of spinal shock is due to the sudden interruption of suprasegmental descending fiber systems that normally keep the spinal motor neurons in a continuous state of readiness

Stage of Increased Reflex Activity

After few weeks of complete transection of spinal cord the reflexes begin to reappear. All the deep tendon reflexes below the level of lesion become exaggerated. Superficial plantar response is extensor with extension of great toe and fanning of other toes. The bladder detrusor becomes hypertonic with spontaneous micturition .Reflex defecation also occurs. After several months the withdrawal reflexes become exaggerated and may be accompanied by profuse sweating, piloerection and automatic emptying of bladder . This is mass reflex which is brought by stimulating the skin of the thigh.

Hemisection of the spinal cord (brown sequard syndrome)

The clinical presentation consists of

1. Ipsilateral loss of proprioceptive function (vibration and position sense) below the level of lesion

2. Contralateral loss of pain and temperature due to the involvement of lateral spinothalamic tract

3. Ipsilateral weakness with spasticity due to the involvement of the corticospinal tract involvement with a segmental lower motor neuron and sensory signs at the level of the lesion

Brown Sequard syndrome is a rare entity and has been described following gun shot injuries.

Central cord syndrome

The pathogenesis in this syndrome starts centrally and spreads to involve the crossing fibres of spinothalamic tract in the anterior commisure resulting suspended or vest like distribution of loss of pain and temperature with preserved touch and proprioceptive sensation known as the dissociated sensory loss. The lesion can extend anteriorly involving the anterior horn cell and may extend laterally involving the pyramidal tract or the spinothalamic tract below the level of lesion.

Central cord syndrome occur acutely due to severe hyperextension injuries of the neck resulting in hematomyelia or chronic lesion such as syringomyelia or intramedullary tumors.[6]

Posterolateral column disease

The lateral column and posterior column involvement result in loss of proprioception and manifest as sensory ataxia with spastic weakness of the limbs. This type of presentation is commonly seen in non compressive lesion due to vitamin B12 deficiency and HIV associated vacuolar myelopathy. [7]

Disease of the anterior horn cell and pyramidal tract

Motor neuron disease such as amyotrophic lateral sclerosis occurs due to degeneration of anterior horn cells and corticospinal tract.[8] They present with both segmental lower motor neuron signs and upper motor neuron signs.

Spinal cord lesions can be classified as intramedullary or extramedullary lesions Under the extramedullary group the lesion can be either intradural or extradural.

Compressive myelopathy

Compression of cord can be extramedullary or intramedullary. Under extramedullary group degenerative disease of the spine is the most common cause. Cervical spondylosis is an important etiological factor in cord compression after the age of 40.

Spondylosis is a term which encompasses degeneration of the ligaments, intervertebral disc, joints and connective tissue of the vertebra.[9] Cervical spondylotic changes are more common in lower cervical region from C4-C7.[10] They usually start in the intervertebral disc as disc dehydration due to loss of proteoglycan matrix. This results in

narrowing of disc space with the formation of vertebral osteophytes ,facet joint hypertrophy and ossification of ligaments.

These changes result in compression of either the cord or root. Cord compression in cervical spondylosis occurs due to three ways.[11,12]

1. static–mechanical compression by the osteophytes, intervertebral disc and spinal ligaments can encroach the spinal canal resulting in canal stenosis. Anteriorly the cord is compressed by vertebral osteophytes,intervertebral disc and ossified posterior longtitudinal ligament. Loss of cervical lordosis worsens the anterior compression since the cord is pushed anteriorly within the spinal canal. Lateral and posteror compression result from vertebral osteophytes and thickened ligamentum flavum.

2. Dynamic compression of cord occurs during neck movements . flexion of the neck result in narrowing of antero posterior diameter of cervical canal by 2-3 mm causing anterior compression of the cord

3. Spondylotic changes can cause an impairment of the circulation within the spinal cord resulting in cord ischemia and myelopathy. Osteophytes can compress either the anterior spinal artery ,

medullary arteries or the venous drainage and cause ischemic myelopathy.

Patients with spondylotic myelopathy present with deep aching or burning pain around the upper extremities(brachialgia). Cervical spondylotic neck pain is felt in the posterior neck region over paraspinal region and also have pain in interscapular and occipital region. Sensory loss develops below the level of lesion with pain and temperature sensation affected earlier than proprioceptive sensation. Patient will have weakness of upper extremity with loss of dexterity followed by lower limb weakness. Unilateral or bilateral weakness depends on the level of compression. Bowel and bladder disturbances result from the involvement of the descending sympathetic and parasympathetic fibers to the bladder.

Cervical spondylotic neck pain is felt in the posterior neck region over paraspinal region and also have pain in interscapular and occipital region. Motor examination reveals the presence of segmental lower motor neuron signs in upper limb with upper motor neuron signs below the level of compression.[13] Hyporeflexia at the segment of compression and hyperreflexia below the level of lesion with or without clonus and extensor plantar response will be present. Flexion of the neck resulting in

painful sensation radiating down the spinal column is known as Lhermite sign and it is seen spondylotic myelopathy. Gait dysfunction occurs due to spastic weakness of both lower limbs

Plain X rays are useful in initial evaluation of spondylotic changes and help in the detection of

1. Disc space narrowing
2. Osteophytes
3. Kyphosis
4. Joint subluxation
5. Stenosis of spinal canal

Computed tomography is better than plain X rays in assessing the canal stenosis , neural foramina narrowing , osteophytes and calcification of ligaments

MRI is the imaging modality of choice in the evaluation of cervical spondylotic myelopathy for the following reasons [14]

1. It accurately measures the spinal canal diameter[15]

2. T2 hyperintensities of spinal cord at the level of cord impingement may be due to ischemia , edema,ischemia ,gliosis and myelomalacia

3. Signal changes of the cord indicate an irreversible damage to the cord

Somatosensory evoked potential in spondylotic myelopathy[16]

The presence of normal cortical P_{20} potential with the absence or reduced amplitude of N_{13} potential indicates compression involving the dorsal gray column of the cervical cord with the sparing of the dorsal column.

The absence or reduced N_{13} potential in median SSEP than in ulnar nerve SSEP helps in localizing the level of compression in C_5 level which correlates with MRI finding[17]

Prolongation of N_{20} latency and the interpeal latency of N_{13} – P_{20}latency indicates dorsal column dysfunction in the cervical cord , medial lemniscus or thalamocortical fiber involvement upto the sensory cortex

Subclinical involvement of the dorsal column with the presence of normal vibration and position sense can be identified by the prolonged interpeak latency of N_{13} –$P_{20.}$

Ossification of the Posterior Longitudinal Ligament

Behind the vertebral body is where the posterior longtitudinal ligament is situated. It extends the entire spinal canal and it separates the spinal cord from the vertebral body and intervertebral disc. Ossification of the posterior longtitudinal ligament is more common in asian population. It is more commonly seen in elderly population secondary to degenerative changes of spine and other causes include fluorosis and hemochromatosis.

Tuberculosis of spine

In developing countries like India the incidence of tuberculosis is still high.[18] Spinal tuberculosis is usually secondary to a infection in the lung or genitourinary system. Spread to the spine is by hematogenous route.Paraspinal abscess consist of necrotic bone matter and breakdown of inflammatory cells. The pus is usually localized or may track down the tissue planes. The infection starts in the anterior aspect of vertebral body adjacent to the disc. Infection spreads to the adjacent vertebral body under the longtitudinal ligaments. Collapse of the vertebral body result in kyphotic deformity

Patterns of Vertebral Involvement

• The primary focus of infection in the spine can be either in the vertebral body or in the posterior elements. Commonest site of involvement is the thoracic spine

• Four patterns are recognised

1. Paradiscal (Commonest)
2. Central
3. Anterior
4. Appendiceal

Paradiscal lesion is the most common site of involvement. Intervertebral disk space narrowing is caused by

1. Destruction of subchondral bone with subsequent herniation of the disc into the vertebral body or

2. By direct involvement of the disc

In Anterior subtype the lesion is subperiosteal in location under the anterior longtitudinal ligament. Pus spreads under the anterior longtitudinal ligament by stripping the periosteum of contiguous vertebra resulting in ischemia of vertebra. Collapse of vertebra and IV disc space narrowing occurs in very late stage of disease.

Bony destruction result in kyphotic deformity known as Potts spine. Kyphosis is divided into two types .[19] knuckle kyphosis occurs when collapse involves one or two vertebra. Angular kyphosis is due to collapse of 3 or more vertebral segments

Tuberculosis of spine with paraplegia

Thoracic spine is the commonest site of involvement. Motor dysfunction occur earlier than loss of sensory functions. Posterior column sensations are last to disappear.

Paraplegia in spinal tuberculosis occurs by various mechanism

1. Inflammatory edema- due to the release of toxins and vascular stasis
2. Extradural compression through epidural abscess
3. Spinal arachnoiditis
4. Bony deformities like kyphosis
5. Vascular insufficiency- endarteritis resulting from inflammation and thrombosis of the vessel
6. Spinal cord changes such as myelomalacia and syringomyelia

Diagnosis

Patients presenting with low back pain , constitutional symptoms with paraplegia and kyphotic bony deformity always suspect tuberculosis of spine.

In plain X rays ,presence of vertebral lesion with end plate irregularity, narrowing of disc space, and paravertebral masses suggestive of epidural abscess ,collapse of vertebra with gibbus formation helps in diagnosing spinal tuberculosis.

Computed tomography (CT) can identify bony sclerosis and destruction within the vertebral bodies. CT is very helpful to guide percutaneous biopsy of infected bone or soft tissue structures

Magnetic resonance imaging helps in [20]

1. Assessing early marrow infiltration of vertebral bodies and discitis
2. Assessment of extradural abscess and subligamentous spread
3. Identifying spinal cord involvement and spinal arachnoiditis
4. Delineates leptomeningeal disease

SSEP studies of tibial nerve help in localizing the level of lesion. The cortical potentials (P_{37}) are of delayed in latency. They can be used as a follow up tool after therapy with ATT[21]

Other causes for compression include spinal cord tumors , vertebral metastasis with cord compression

Intraspinal tumors

Spinal cord can be compressed by tumors arising within the substance of spinal cord(intramedullary) or from outside the cord(extramedullary) such as vertebral bodies (extradural) or leptomeninges(intradural)

The incidence of spinal tumors in the descending order of frequency are extradural(55%) followed by intradural(40%) and intramedullary(5%). In extramedullary tumors, meningiomas and neurofibromas are the most common . These tumors are intradural. Neurofibromas are more common in the thoracic and lumbar region. Meningiomas most commonly involve the vertical extent of the spinal cord.

In the intramedullary subset of tumors, ependymomas and astrocytoma are common. Ependymomas constitute 60% followed by astroctomas (25%).

NON COMPRESSIVE MYELOPATHY

They form a major subset of myelopathies and can be subdivided based on infections, demyelinating, vascular, toxin and degenarative disease. In India nutritional and infections forms a major cause of non compressive myelopathy in contrast to western population where demyelination and degenerative causes are more common

Acute transverse myelitis (ATM)

Inflammation of the spinal cord present acutely with features of complete transection of the cord. They represent a major subset in non compressive myelopathies.

Acute transverse myelitis usually occur in post vaccinial or postinfectious acute disseminated encephalo myelitis(ADEM), multiple sclerosis, or Neuromyelitis optica.[22] They also occur in association with many rheumatological disorders, vascular and paraneoplastic syndrome. If the cause for myelitis is not known and if the lesion is monophasic it is usually termed as idiopathic transverse myelitis.

Idiopathic transverse myelitis is diagnosed when all of the inclusion criteria is present and all of secondary causes are excluded.[23] Disease associated ATM should fulfill all of the inclusion criteria and the patient should have an underlying secondary condition mentioned in the exclusion criteria

The thoracic cord is the most common site of involvement in acute transverse myelitis. Neuroimaging with magnetic resonance imaging shows the following features [24]

1. Cord enlargement in sagittal T1 sequences
2. Presence of T2 hyperintensities in three or four segments
3. Involvement of more than two thirds of the cord in axial T2 sequences
4. On contrast administration there is a peripheral rim of enhancement

In multiple sclerosis , the lesion involves one or two segments of the cord, occupies the periphery of the cord and with contrast there is enhancement at the center.

SSEPs in demyelinating diseases

In multiple sclerosis SSEP has a role in detecting clinically silent lesions. Central sensory conduction time is prolonged due to temporal dispersion. As a result there is delay in the cortical responses and prolongation of interpeak latencies. SSEPs is superior to other evoked potentials such as visual evoked potential(VEP) and Brainstem auditory evoked potential(BAER).The sensitivity of SSEPs is greater than other evoked potentials due to the greater length of the somatosensory pathways when compared to visual and auditory pathways. The yield of tibial SSEPs is more with tibial nerve when compared to median nerve. A significant correlation is found between the prolongation of cervical(N13) and coprtical(N20) potential following median nerve stimulation and the MRI evidence of signal changes in posterior column of cervical cord.

Acute transverse myelitis , most commonly involve the thoracic cord and hence median SSEPs is normal. Tibial SSEPs correlates with sensory findings.[25] Follow up of patients with serial Tibial SSEPs showed persistence of abnormalities. Hence tibial SSEPs is used to prognosticate the disease

SSEP studies may help in patients where MRI is normal with CSF evidence of elevated protein indicating inflammation of cord. In follow up studies SSEPs may help in assessment of prognosis of the lesion.

Subacute combined degeneration (SACD) of the spinal cord

Deficiency of vitamin B12 can present as a multisystem disorder affecting the central and peripheral nervous system with psychiatric and hematological involvement. They have a spectrum of neurologic presentation including neuropathy, myelopathy, optic neuropathy or cognitive impairment.

Cyanocobalamine is necessary for the conversion of methylmalonyl-coenzyme A to succinyl-coenzyme A and also for conversion of homocysteine to methionine.Defect in the synthesis of methionine may lead to depletion of S-adenosylmethionine which is necessary for the synthesis of myelin phospholipids. Impairment in the production of succinyl Co A result in production of odd chain fatty acids which get incorporated into the myelin resulting in abnormal conduction.

The clinical manifestation of SACD include presence of paresthesia of feet with gait disturbances such as unsteadiness getting more aggravated during eye closure. On clinical examination there is early loss of vibration and position sense in the lower limbs with spastic

paraperesis and extensor plantar response.[26] Knee and ankle jerks are exaggerated but in the presence of neuropathy ankle jerks are absent.

Peripheral smear and bone marrow examination may show the presence of macrocytosis and hypersegmented polymorphonuclear cells .The presence of low serum B12 levels and elevated homocysteine and methyl malonic acid help in the diagnosis.

MRI spine can demonstrate T2 hyperintensities of posterior and lateral column but it is not seen in all patients.[27] SSEPs can detect subclinical B12 deficiency. There is a prolongation of central conduction time . Median and tibial SSEPs showed prolonged latencies of cortical responses.[28] After therapy SSEPs showed improvement in the latency of P20 and P37 of median and tibial SSEPs.[29]

HIV associated myelopathy

It usually occurs in late stages of HIV infection when the CD4 counts are very low. Pathologic examination shows the vacuolar changes of the myelin with lipid laden macrophages in the posterior and lateral column with axonal sparing.[30]

They present with paresthesia and numbness of feet , difficulty in walking with sensory ataxia and loss of vibration and position sense of both feet with no definite sensory level and spastic weakness with hyperreflexia of lower limbs.Presence of bladder dysfunction with urgency and urge incontinence is common[31]

MRI of the spine should be done to rule out opportunistic infection of the spinal cord and T2 hyperintensities may be seen in posterior column of spinal cord.[32]

HIV patients develop myelopathy during late stage of infection or due to oppurtunistic infection. SSEPs abnormalities has been noted in 85.7% patients with HIV but only 50% had objective sensory findings.[33] Presence of abnormal SSEPs findings helps in predicting future development of myelopathy. SSEP findings show prolonged latencies of cortical N20 and P37 responses.Treatment with antiretroviral therapy (HAART) may improve the symptoms.

Other non compressive myelopathy include degenerative diseases such as hereditary spastic paraplegia,and amyotrophic Lateral sclerosis,

In Hereditary Spastic Paraplegia(HSP), pure form is characterized by spastic paraperesis with autosommal dominant pattern of inheritance manifesting in the second or third decade[34]

Complicated HSP includes the presence of peripheral neuropathy, seizures,dementia , ataxia, optic atrophy and sensori neural hearing loss. Histopathological evaluation reveals axonal degeneration involving ascending and descending tracts with sparing of nerve roots and peripheral nerves.

MRI of spine is usually normal at times may be associated with atrophy of corpus callosum.[35] Though by clinical examination posterior column functions may be normal, tibial SSEP shows reduced amplitude or delayed cortical(P37) response.[36]

MATERIALS AND METHODS

The study was conducted on patients attending neurology services at the Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital from April 2010 January 2012.The approval from the Institutional ethical Committee and informed consent from the patients participating in the study were obtained.

Fifty two patients with clinical and radiological features of myelopathy were included in this study and were subdivided into two groups of compressive and non compressive myelopathy. They were compared with age and sex matched controls.

Exclusion criteria :

Patients with coexisting neuropathy, radiculopathy , brainstem or cerebral cortical lesions were excluded from the study since they had a confounding effect on the somatosensory evoked potential (SSEP) test result.

Patients who have systemic disease such as diabetes mellitus , hypothyroidism,systemic malignancy, chronic drug or alcohol intake and connective tissue disease such as systemic lupus erythematosus or

sjogren syndrome were excluded since they may affect the peripheral nerve conduction.

All patients underwent a detailed neurological examination.

Blood investigations such as complete hemogram, blood urea , sugar, serum creatinine, HIV, VDRL, chest x-ray and spine xray were done.

In patients with non compressive myelopathy blood for vitB12 levels, CSF analysis for cell count, cytology , protein and oligoclonal bands were done. Detailed rheumatological work up were done for all patients with acute transverse myelitis since secondary causes has to be ruled out.

Magnetic resonance imaging(MRI) of the spine with or without contrast and brain screening was done with 1.5 Tesla MRI . The following sequences were done,

- T1 and T2 sagittal
- Post gadolinium T1 contrast
- T2 axial

Electrophysiological study :

Median and tibial motor and F wave,median and sural sensory conduction studies were done bilaterally to exclude peripheral neuropathy. Tibial and median SSEP were done bilaterally.

Somatosensory evoked potential (SSEP)

Introduction:

Somatosensory evoked potential (SSEP) are electrical potentials generated mainly by large diameter sensory fibers in the central and peripheral nervous system following a stimulus. There are three categories of potentials based on the latency short latency(within 50 milliseconds), middle latency(50-100 ms) and long latency(100-300ms). But short latency potentials are routinely analysed in clinical practice because the middle and long latency potentials are inconsistent and variable.

Physiology of SSEP:

The proprioceptive pathways are mainly assessed by the SEP studies.

After an electrical stimulation impulses travel via myelinated type A fibers. The first order neurons are located in the dorsal root ganglia.

The impulses ascend via the medial division of the root and the fasciculus gracilis and cuneatus in the dorsal column of spinal cord. The second order neurons arise from the nucleus of gracile and cuneatus and cross to the opposite side and ascend in the medial lemniscus to the ventropostero lateral nucleus of thalamus. The thalamoparietal radiations enter the posterior limb of internal capsule and terminate in the primary sensory cortex.

Usefullness of SSEP in disorders of central nervous system

SEP studies are helpful in localising lesions along the central somatosensory pathways. Following are the disease states in which SEP has a definite role

Multiple sclerosis(MS):

SEP studies help in identifying early subclinical involvement of the central somatosensory pathaways. Among the evoked potentials SEPs are superior in identifying subclinical lesions. SEP abnormalities in MS has a diagnostic relevance by detecting and localizing multiple lesions of the cord and brain.

HIV and Vitamin B 12deficiency myelopathy present with posterior column and pyramidal tract dysfunction. Magnetic resonance imaging may reveal signal changes in the posterior spinal cord region. In few cases MRI may be normal and for these patients SSEP help in identifying the lesion in the cord

Cervical spondylosis

Presence of both radiculopathy and myelopathy can affect SSEP potentials. SSEP are used in the follow up of patients undergoing surgery to monitor the effect of treatment. Absence of SEPs potentials even months after surgery have a poor prognosis.

Tumors of spinal cord

SSEPs can be abnormal in both intramedullary and extramedullary tumors. Intraoperative monitoring of SSEPs can help the surgeon in identifying the functional continuity of nerve roots in the spinal cord. It helps in reducing the postoperative neurological deficits by around 50%.

Methods

Pretest instructions

On the day of procedure patients are advised to take head bath with no oil application to the scalp.The test is done with patient in supine

position with neck muscles relaxed by a proper head support. The patient should be relaxed and comfortable to avoid muscle artifacts.

Machine settings

The SSEPs are best recorded by amplification of the potentials between 10,000- 5,00,000. The impedance was kept below 5Ω. The filter setting was 20-30 Hz for low filters and 3000Hz for high filters. Analysis time base should be 50-60ms which can be extended to 60-100 ms. In the latter setting low filter should be set at 1-3 Hz and 1000-2000 responses should be averaged. The skin was thoroughly cleaned with alcohol to reduce artifacts. For median SSEPs the stimulus was applied 2 cm proximal to skin crease .

Stimulus factors

A 200μV square wave pulse sufficient to produce a painless twitch of the thumb in median SSEPs and the toes in tibial SSEPs was applied. A current ranging in amplitude from 5-15 mv was used for stimulation. The rate of stimulation was between 3Hz and 8Hz.High rate stimulation is painful and results in progressive loss of amplitude and decrease in latency. Since the SEP waveforms are very small, 1000 or more epochs were averaged.

Median SSEP

Position of the electrodes

1cm disc electrodes filled with appropriate conducting jelly or paste was used . Stimulation is given to the wrist with the cathode 2 cm proximal to the skin crease The electrodes were placed in the following position for right sided median nerve stimulation

i. Erb ′s point (the electrodes were placed 2-3 cm above the midpoint of clavicle) on the right(ipsilateral) side

ii. Spinous process of the fifth cervical vertebra

iii. 2cm posterior to C3 position(contra lateral scalp) of 10-20 international system of EEG electrode placement(C3′)

The spinal electrode is designated as C_5S or C_5Sp, and Erb s point electrodes EP_1 and EP_2 were assigned to left and right side. The montage for median SSEP is as follows

Channel 1:$C_{3/4}$-Fz

Channel 2:EP_1/EP_2-Fz

Channel 3:C_5Sp-Fz

Channel 4: antecubital fossa

Median SSEP waveforms:

Erb ′s potential(N_9) is seen as a negative peak in EP-Fz channel. Spinal potential (N13) is a negative peak seen in C_5Sp-Fz. The N_{20} waveform is also a negative peak seen in $C_{3/4}$- Fz channel and is followed by a positive cortical potential $P_{25.}$

The site of origin of these different waveforms is unique. The N_9 peak originates in the distal brachial plexus .N_{13} peak originates in the dorsal grey horn of cervical cord. The amplitude of N_{13} is maximum between C3- C7 and it is not recordable above C2. N_{20} potential is thought to originate from the contralateral somatosensory cortex in the posterior lip of central fissure.

The waveforms are analysed for following parameters

1. latency

2. amplitude

3. interpeak latency

The latency is measured from the stimulus artifact to the peak of waveform and two inter peak latencies are calculated

1.Brachial plexus to the spinal cord (N_9-N_{13})

2.Central sensory conduction time(N_{13}-N_{20})

Amplitude of wave forms are less important in clinical practice since their values are variable and do not follow a normal distribution.

Tibial SSEP

Procedure

The posterior tibial nerve is stimulated at the ankle with the cathode between the medial malleolus and tendoachilles tendon and the anode 3 cm distal to the cathode. A 200-300µs square wave electrical pulse with a minimum current to produce a painless visible twitch of the toes were delivered at the rate of 3-8 Hz. The filter settings are similar to median SSEP.

Placement of electrodes

In the popliteal fossa recording electrode is placed 4-6cm above the popliteal crease midway between the semitendinous and biceps femoris with reference electrode on the medial surface of knee. Spinal electrodes are placed over the T12 vertebra with referrence to iliac crest and over C_5Sp which is referred to Fz. The scalp recording electrode is placed 2cm posterior to Cz(Cz′) referred to Fz.

For evaluation of central somatosensory pathway following montage is used

Channel 1:Cz′-Fz

Channel 2:C_5SP-fz

Channel 3:T_{12}-iliac crest

Channel 4:popliteal fossa(PF)- knee(K)

Tibial SSEP waveforms

The N_7 is the negative potential recorded in the popliteal fossa - knee channel.N_{20} is the predominant negative peak obtained in the T12 – iliac crest channel. In C_5Sp –Fz channel a negative peak (N_{28}) is seen at 28 milliseconds. The cortical potential P37 is seen as a major positive peak in the Cz′-Fz channel.

Generators of tibial SSEP

N_7 potential originates in the tibial nerve. The N_{20} potential probably originates from the dorsal gray matter of the lumbar spinal cord. N_{28} potential arises from the dorsal gray column of the cervical cord. P_{37} potential originates from the scalp region overlying the primary sensory area of the leg.

The latencies and inter peak latencies are measured similar to median SSEP. The interpeak latency N_{20}- P_{37} measures the central conduction from the lumbar cord upto the sensory cortex.

OBSERVATION AND RESULTS

The study included fifty two patients with clinical features of myelopathy of which twenty six patients were in the compressive group and twenty six were in the non compressive group. There were 35 males and and 17 females .

Among 26 patients in the compressive myelopathy, 19 were male and 7 were female .Age of the patients ranged between 24-73years. The mean age was 53.07 years. In the non compressive myelopathy , 16 were male and 10 were female. The age of the patients ranged between 24-48years.The mean age was 35.15 years.

Control population included 20 patients with 13 male and 7 female aged between 18-65 years.The mean age was 36.45 years. Median and tibial SSEP were done bilaterally for the control population and the normal values are given below

Median SSEP

Potential	Mean latency	SD	UL-MEAN+3SD
N9	8.91	0.398	10.1
N13	13.29	0.53	14.89
N20	20.61	0.83	23.12
N13-N20	7.33	0.77	9.67

Tibial SSEP

Potential	Mean latency	SD	UL-MEAN+3SD
N7	7.23	O.46	8.63
N20	20.90	0.97	23.83
N28	28.33	0.79	30.7
P37	38.18	0.88	40.83
N20-N28	7.42	0.90	10.13
N20-P37	17.28	1.01	20.31

COMPRESSIVE MYELOPATHY

Cervical spondylosis was the most common etiology of compressive myelopathy.Among them cord compression was most common in C5 and C6 segments.The second common etiology was tuberculosis of the spine and in those patients thoracic vertebra was the common site of involvement.

Table 1.Etiology Of Compressive Myelopathy

Etiology	No. of.patients(N=26)	Percentage(%)
Cervical spondylosis	18	69.23
Spinal tuberculosis	3	11.53
OPLL	3	11.53
Disc herniation	1	3.84
Secondaries of spine	1	3.84

OPLL-ossification of posterior longtitudinal ligament

Table2. Age wise distribution of cervical spondylotic myelopathy(including disc herniation)

Age group	No.of patients(n=19)	Percentage(%)
50-60years	2	10.52
60-70years	10	52.63
70-80years	7	36.84

Cervical spondylosis is usually due to the degeneration of spine and becomes symptomatic after fifth decade. In our study it was more common in the seventh decade .

Table 3 Cord Compression In Degenarative Disease Of Spine

Cord level	No of patients(n=22)	Percentage
C4	3	13.63
C5	11	50
C6	5	22.72
C7	3	13.63

Degenarative disease include cervical spondylosis, OPLL,and disc herniation

Twenty two(84.61%) patients with degenerative disease of cervical spine presented with quadriparesis and brisk tendon reflexes below the level of compression. Posterior column dysfunction was present in 13 (59.09%) , spinothalamic tract dysfunction was seen in 20(90.90%) and bladder symptoms were present in 6(27.27 %) patients in the cervical spondylotic myelopathy and OPLL group .

MRI demonstrated disc protrusion with narrowing of thecal space, and T2 signal hyperintensities in 15 (84.21%) patients. Cervical canal stenosis due to OPLL was seen in three patients. OPLL was confirmed by CT scan of the spine.

N13 potential of the median SSEP was prolonged in sixteen(84.21%) cervical spondylosis cases with compression of C5,C6

segments. N13 potential was normal in three cases(15.78%) of C7 cord compression.Interpeak latency of N13- N20 were prolonged in fifteen patients of compressive myelopathy. Delayed or absent N13 potential correlated well with absent tendon reflexes in the corresponding upper limb(x^2,$p<.05$). N13-N20 interpeak latency was prolonged when the compression was at and above C5. Posterior column sensations were present in six out of sixteen patients with cord compression at C5,C6 segments . There was no significant correlation between the N13 potential and posterior column sensations (p -0.696)

Tuberculosis of spine was diagnosed in three(11.53%) patients and all were females. The age of patients were between 20-40 years. They all presented with low back pain and paraperesis. Posterior column,spinothalamic and bladder dysfunction was present in all three patients . Xray showed involvement of dorsal vertebra with adjacent inter vertebral(IV) disc space narrowing and gibbus in three patients .MRI showed vertebral end plate signal changes in D5, D6 vertebra in two patients . IV disc space narrowing and gibbus was demonstrated in all three patients .

Median SSEPs showed normal N13, N20 latencies and central conduction time(N13-N20) . They all had delayed P37 potential and prolonged central conduction time(N20-N28) in tibial SSEPs

One patient with D10 vertebral metastasis had clinical and electrophysiology features similar to spinal tuberculosis. In MRI lytic lesions of bone with normal IV disc space and preserved vertebral end plate was seen.

NON COMPRESSIVE MYELOPATHY

In 26 patients of non compressive myelopathy 17 had acute transverse myelitis(65.38%) and it was the commonest cause among the non compressive myelopathies.

Table3. Etiology Of Non Compressive Myelopathy

Disease	No of patients	Percentage of patients
Acute transverse myelitis	17	65.38
Multiple sclerosis	3	11.53
Hereditary spastic paraplegia	3	11.53
SACD	2	7.69
HIV associated myelopathy	1	3.84

SACD- Subacute Combined Degenaration of Cord

The age of patients ranged between 24-48 years and mean age was 35.64 years. Among them 12(70.58%) were male and 5(29.41%) were female Fifteen patients (90%) with ATM presented with paraperesis , while two (10%) had quadriparesis.Hyperactive tendon reflexes and spasticity was seen below the level of lesion. Definite sensory level with loss of all modalities of sensation below the level of lesion and bladder dysfunction was seen in all patients.

Definite sensory level were present in 20 patients and out of which 17 had acute transverse myelitis and 3 had multiple sclerosis MRI showed T2 hyperintense signal changes in all patients of ATM and multiple sclerosis and normal in the rest of non compressive myelopathy patients. Thoracic cord involvement was the commonest and seen in 18 patients(90%) followed by the cervical cord in 2 patients(10%) in demyelinating myelopathies.

MRI of ATM in our patients demonstrated T2 hyperintense signal in 3 or more segments of spinal cord with three patients showing a maximum of 6 segments . In seventeen patients with transverse myelitis , thoracic cord was involved in fifteen patients and cervical cord in two patients All lesions were centrally located involving more than 50% of cross section area of the cord and all were contrast enhancing

The spinal cord involvement was less than three segments in all three patients with multiple sclerosis. The hyperintense signal changes were located in the periphery of the cord with all lesions enhancing with the contrast

Optic neuritis was present in three patients(15%) of the transverse myelopathy group . Two patients had bilateral optic neuritis and long segment demyelination involving more than 5 spinal cord segments. Neuromyelitis optica were suspected in those patients but aquaporin antibody assay could not be done.

Cerebrospinal fluid(CSF) examination showed elevated proteins and oligoclonal bands were negative in all the patients with transverse myelitis

Median SSEPs studies including N13, N20, N13-20 were normal and tibial SSEPs showed prolongation of latency of P37 and central conduction time(N20-N28) from the lumbar to cervical cord in patients with thoracic myelopthy.In cervical cord lesion median N13 and N13-20 was prolonged. In our patients with multiple sclerosis thoracic cord was involved in two patients and cervical cord in one patient.SSEPs abnormalities were similar when compared to ATM

SACD or subcacute combined degeneration of cord was seen in two patients.The age of these patients were 34 and 42 years respectively. Both of them were male patients.They had spasticity of both lower limbs with hyper reflexia and paraperesis. Posterior column sensations (vibration and joint position sense) was impaired in both lower limbs. MRI spine was normal. Median nerve SSEPS were normal and tibial SSEPs showed prolongation of P37 cortical response and central conduction time (N20-N28).There was no significance between radiological and SEP abnormalities (p-0.220)

Hereditary spastic paraplegia was seen in three patients(11.53).The age group ranged from 34-42 years. Mean age was 37 years All had spasticity and hyperreflexia of both lower limbs.There was no sensory, bladder, or bowel disturbances. MRI of spine in all three were normal . Median SSEP revealed no abnormality while tibial SSEPs showed prolonged latency of P37 cortical potential and central conduction time(N20-P37).There was no significant association between SSEPs and MRI.

DISCUSSION

Diseases of the spinal cord encompasses a wide range of etiology from compressive to non compressive myelopathy.

Compressive myelopathy

Cervical spondylosis is due to degeneration of the spine and forms the commonest cause of spinal cord disease in elderly population.In our patients with spondylotic myelopathy , 89.5% were above 60 years. Hiyashi et al reported 80.5% patients with spondylotic myelopathy above 60 years. It constituted 84.61% cases in the compressive group including OPLL and disc herniation.. Among them 7 (31.81%)were females and 15(68.18%) were males Cord compression was more common in C5segment than C6 and C7. Hochman et al reported compression to be equally common from C5-C7 levels. [37]

Median N13 potential has good localizing value . The latency of N13 was prolonged in C5 and C6 cord compression. Cord compression below C7 myelopathy had normal N13 potential. Prolongation of N13 latency correlated well with absent tendon reflexes of C5 and C6 segments($p<0.05$). Restuccia et al reported prolonged N13 potentials with C5 myelopathy due to involvement of dorsal grey matter.[16] And this was associated with absent or reduced tendon reflexes of C5 or C6

segment (x^2, $p < 0.05$) similar to our study. He concluded that prolonged or delayed cortical N20 or N13-N20 interval indicates delayed conduction in the somatosensory pathways of dorsal column due to compression.

Tuberculosis of the spine accounted for 11.53 % of cases in the compressive group. It was the second most common cause of compression in our study. In contrast Misra et al in his study reported tuberculosis of spine as the commonest cause of compression (35.71%) followed by cervical spondylotic myelopathy(34.13%).[38] The age of all the three patients ranged between 20-40 years and all were female. Thoracic vertebra was affected in all three patients with D6 vertebra being involved in two patients and D4 vertebra in one patient. Owalabi et al found 53.9% of patients between 20-40years.[39] Among the eighty seven patients 57 were males and 30 were females. Commonest age group was between 30-40 years(40.62%). Thoracic vertebra was the commonest site of involvement(56.7%) of which upper thoracic vertebra was involved frequently followed by lumbar vertebra 28.4%. In contrast Chung et al found lower thoracic vertebra as the commonest site of involvement . Misra et al found thoracic vertebra to be the commonest site(80%) followed by lumbar(13.33%) and cervical (6.7%). In his study the disease most commonly occurred in 2nd and 3rd decade and males were commonly affected(53.3%) than females(45.6%). In our patients

Median SSEPs were normal but tibial SSEPs showed prolonged cortical P37 and N20-N28 interval in all three patients and similar observation was noted by Titlic et al.[21] In his patients with back pain and radiological features of caries spine without neurological deficits also had prolonged P37 and N20-N28interval indicating subclinical cord involvement. He reported that tibial SSEPs could be of use in monitoring response to therapy.

In one patient who had D10 vertebral metastasis clinical ,radiological and electrophysiological correlation was present

Non compressive myelopathy

In our study 26 patients were included in the non compressive group, 16 were males and 10 were females. Mean age at presentation was 35.15 years. ATM formed the major bulk of the non compressive myelopathies accounting for 65.38%.The age of the patients ranged from 24-49 years and the mean age is 35.64years.All ATM s were idiopathic . No secondary causes were found. Prabhakar et al analysed 57 patients of non compressive myelopathy with 42 males and 15 females . Patients age ranged between (14-82years) and the mean age was 34.45 years. He found 54.38% of acute transverse myelitis(ATM) among the non compressive group. Mean age of presentation in ATM was30.35 years (age range 14-65 years)

Fifteen patients (90%) with ATM presented with paraperesis , while two (10%) had quadriparesis. All had a definite sensory loss with loss of all modalities of sensation. Saleh et al analysed thirty one patients with features of ATM , 11 had quadriparesis,20 had paraperesis and all had a definite sensory loss below the level of lesion[40]

Myelopathy due to multiple sclerosis was diagnosed based on history, relapsing and remitting course and MRimaging. In our study 3 patients had multiple sclerosis.Mean age was 32 years and age ranged between(24-42years). Two were males and one female. Two patients presented with complete transverse melopathy while one patient presented with partial cord syndrome with sparing of spinothalamic tract similar to study by Prabakar et al . Oligoclonal bands were negative in all patients

In our study the thoracic cord was the most common site of involvement (88.23%) ,and next was the cervical cord(11.76%). Prabhakar et al in his series demonstrated the location of lesions in MRI, to be in dorsal spine (32%), cervical spine (26%), cervicodorsal (23%), entire cord (3%) and in the conus in (3%).[41] Alper et al demonstrated that in ATM patients cervicothoracic cord was the most common site of involvement.

The MRI spine of all our patients with ATM demonstrated the involvement of more than three segments of the cord, more than 2/3rd the crossectional area of the spine and lesions were centrally located.Bakshi et al demonstrated hyperintense T2 signal changes in more than three segment of cord upto a maximum of six cervical segments.[42]MRI spine of patients with MS in our study showed T2 hyperintense signal changes in less than or equal to three segments of spinal cord which were located in the periphery of the cord similar to the observation by Tartaglino et al.[43]

SSEPs studies in 18 patients with thoracic cord involvement showed normal median SSEPs potentials while tibial SSEPs were abnormal. Latency of P37 potential was delayed and the interpeak latency of N20-N28 and N20-P37 were prolonged. Two of the patients with cervical cord involvement had median SSEPs abnormalities .N13 and N13-20 latency were prolonged. In tibial SSEPs, P37 potential was delayed but N20-N28 interpeak latency was normal . Our observations were similar to the SSEPs analysis by Saleh et al.[40]

Subacute combined degeneration of the cord due to B12 deficiency was seen in two patients. They had spastic paraperesis with posterior column dysfunction and low serum B12 Level. Spinal MRI was normal in these patients Tibial SSEPs showed prolonged cortical p37

latency and central conduction time (N20-N28) which was significant($p<.05, x^2$).Following therapy with vitamin B12 ,P37 potential of tibial SEP was still prolonged after 6 months but the patient had clinically improved . Hemmer et al in his series presented nine patients, six were female and three were male.[44] The clinical presentation was similar to our patients except 4 patients had absent ankle jerks suggestive of neuropathy and one had a subcortical cognitive dysfunction. MRI spine was normal in two out of his seven patients and in all the patients tibial SSEP showed delayed cortical P37 potential and prolonged central conduction time in the cord.Six months after treatment with vitamin B12 , the cortical potential(p37) was still prolonged**.**

One patient with HIV myelopathy had clinical , MRI and electrophysiological findings similar to B12 deficiency myelopathy.Fernandez et al reported a HIV myelopathy with MRI showing hyperintensity in posterior column of thoracic cord and prolonged P37 latency of tibial SSEP.[45]

Hereditary spastic paraplegia was seen in three patients. Clinically all had spastic paraperesis with no sensory and bladder involvement.MRI spine and median SSEP were normal . But tibial SEP showed prolonged

P37 waveform and N2O-P37 latencies.Luciana et al analysed 11 patients with HSP. The clinical features were similar to our patients.[36] Out of which five patients had prolonged latency of cortical P37 potential and central conduction time(N20-P37). He found that the longer the pathway the higher the incidence and severity of the electrophysiological abnormalities and hence tibial SSEP were abnormal rather than median SSEP

CONCLUSION

- Cervical spondylotic myelopathy was the commonest cause for cord compression .C5 and C6 segments were frequently involved

- Absent or prolonged latencies of N13 potential in median SSEP correlated with absent or reduced tendon reflexes of C5 and C6 segment

- In patients with cervical spondylotic myelopathy(CSM) who had intact posterior column sensations median SSEP´ s were abnormal indicating subclinical involvement and hence intraoperative monitoring may be helpful during surgery for CSM.

- Tuberculosis of spine involving the dorsal vertebra is associated with delayed cortical P37 potential and prolonged N20-N28 interval of tibial SSEPs.They can be used in follow up of patients without gross neurological deficits after instituition of antituberculous treatment

- In degenerative disease such as hereditary spastic paraplegia , abnormalities were seen in tibial SSEPs indicating that the degeneration is not restricted to motor pathways but also involves the central sensory pathways

- In patients with subacute combined degeneration of the cord and HIV myelopathy having normal MRI, posterior column involvement is indicated by abnormal SSEPs .Tibial SSEPs may not be of use in monitoring therapy in SACD as they may be abnormal even though patient has clinically improved

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PROFORMA

Name age sex MIN

Address

Presenting complaints

History of present illness

Motor weakness:

Sensory disturbances:

Bladder/bowel disturbance:

Erectile dysfunction

Trophic ulcers,nail ,hair changes

Radicular/funicular pain

Cranial n disturbance

Higher mental function disturbance:

past h/o: connective tissue disease, pulmonary tuberculosis,recent vaccination , Dog bite

h/o drug/toxin exposure/Trauma

personal h/o: alcohol/tobacco/exposure to CSW

Family h/o similar illness:

On examination

General examination:

B.P: PR: MMSE:

Cranial nerve:

Spinomotor system

Bulk tone

Power

UL LL

Reflex-sup DTR

sensory

Touch,position and vibration

Pain/Temperature

Cerebellum

EPS

Investigation

Blood

Sugar: urea: sr.creatinine

Hb - PCV- TC: DC: ESR:

ELISA HIV

Blood VDRL:

serum B 12

CSF:protein/sugar/OCB/cell count

MRI of spine

Nerve conduction study

nerve	Distal latency	Amplitude distal	Amplitude proximal	Conduction velocity	F min
Median –right					
Median –left					
Tibial- right					
Tibial -left					

Median SSEP

Potential	latency	Interpeak latency
N9		
N13		
N20		
N13-20		

Tibial SSEP

Potential	Latency	Interpeak latency
N7		
N20		
N28		
P37		
N20-28		
N20-P37		

ABBREVIATIONS

SSEP- somatosensory evoked potential

HSP- Herditary Spastic Paraplegia

SACD-Subacute combined degeneration of cord

ATM- Acute Transverse Myelitis

CSM-Cervical Spondylotic Myelopathy

OPLL- Ossification Of Posterior Longtitudinal Ligament

MRI-Magnetic Resonance Imaging

	CM																						
Sl.No.	NAME		sex	age	Age Rg		reflex.L	sensory L	PAIN	P&V	BS	MRI	Mrsignal	C3	C4	C5	C6	C7	D1-6	D7-12			
1	karnan		1	55	4		1	1	0	1	0	1		1	1	0	0	0	0	0			
2	balachandran		1	73	6		1	1	0	1	1	1		1	1	0	0	0	0	0			
3	balaji		1	46	3		2	2	0	0	0	1		0	0	1	0	0	0	0			
4	Devaraj		1	52	4		8	3	0	0	0	1		0	0	1	0	0	0	0			
5	Rahul		1	65	5		4	4	0	0	1	1		0	0	0	0	1	0	0			
6	Vanaja		2	62	5		2	2	0	1	0	1		0	0	1	0	0	0	0			
7	kavitha		2	55	4		2	2	0	0	0	1		0	0	1	0	0	0	0			
8	Rajndran		1	48	3		2	2	0	1	0	1		0	0	1	0	0	0	0			
9	vijayan		1	56	4		4	4	0	0	0	1		0	0	0	0	1	0	0			
10	mary		2	35	2		3	3	0	0	1	1		0	0	0	1	0	0	0			
11	Senthil		1	68	5		1	1	0	0	1	1		1	1	0	0	0	0	0			
12	venkatesh		1	64	5		2	2	0	0	0	1		0	0	1	0	0	0	0			
13	ramesh		1	53	4		2	2	0	0	0	1		0	0	1	1	0	0	0			
14	john		1	58	4		2	2	0	0	0	1		0	0	1	1	0	0	0			
15	Vadivel		1	63	5		4	4	1	1	1	1		0	0	0	0	1	0	0			
16	RAMU		1	45	4		2	2	0	0	0	1		0	0	1	0	0	0	0			
17	jothi		2	47	4		2	2	0	1	0	1		0	0	1	0	0	0	0			
18	meena		2	34	2		7	5	0	0	1	2		0	0	0	0	0	1	0			
19	krishnan		2	28	1		7	5	0	0	0	2		0	0	0	0	0	1	0			
20	lalitha		2	24	1		7	5	0	0	0	2		0	0	0	0	0	1	0			
21	mani		1	54	4		3	3	1	1	0	1		0	0	0	1	0	0	0			
22	girija		2	56	4		3	3	0	1	0	1		0	0	0	1	0	0	0			
23	jeyalakshmi		2	63	5		3	3	0	0	0	1		0	0	0	1	0	0	0			
24	selvi		2	59	4		2	2	0	1	0	1		0	0	1	0	0	0	0			
25	RAVI		1	55	4		2	2	0	0	0	1		0	0	1	1	0	0	0			
26	vasudevan		1	62	5		5	6	0	0	1	2		0	0	0	0	0	0	1			

COMPRESSIVE MYELOPATHY GROUP																							
Sl.No.	Median Right					Median Left					Tibial Right							Tibial Left					
	N9	N13	N20	N13-20		N9	N13	N20	N13-20		N7	N20	N28	P37	N20-N28	N20-P37		N7	N20	N28	P37	N20-N28	N20-P37
1	9.2	14	24.2	10.7		9.3	13.8	24.2	10.4		7.3	21.2	28.3	40.4	7.1	19.2		7.5	21.8	29.3	40.5	7.5	18.7
2	8.7	14	23.8	9.6		8.9	13.5	24.8	11.3		8.2	22.1	28.7	40.7	6.6	18.6		8.3	22.4	28.9	40.9	6.5	18.5
3	8.9	17	26.3	9.8		8.6	16.8	25.2	8.4		7.6	21.8	30.5	42.4	8.7	20.6		7.8	21.9	31.4	41.7	9.5	19.8
4	9.2	17	26.3	9.5		9.3	16.3	25.8	9.5		7.4	22.3	31.5	41.8	9.2	19.5		7.5	22.2	32.2	41.7	10	19.5
5	9.1	14	20.2	6.7		9.2	13.7	20.5	6.8		7.8	22.6	32.2	42.32	9.6	19.72		7.9	21.7	32.9	42.5	11.2	20.8
6	9.4	17	27.2	10.3		9.5	16.8	26.9	10.1		7.5	21.6	31.8	42.4	10.2	20.8		7.2	22.3	31.9	42.5	9.6	20.2
7	9.5	17	26.9	9.7		8.9	16.5	26.8	10.3		6.8	22.1	31.5	42.5	9.7	20.4		6.9	21.8	32.2	42.1	10.4	20.3
8	8.8	16	25.6	10.1		9.3	15.8	26.1	10.3		7.1	21.7	31.1	42	9.4	20.3		7.2	21.2	31.8	41.6	10.6	20.4
9	8.9	14	20.1	6.3		9.2	13.9	21.4	7.5		7.5	21.5	32.5	42.4	11	20.9		7.8	21.3	31.2	41.9	9.9	20.6
10	9.3	16	25.8	10.1		9.2	15.9	25.8	9.9		7.2	20.9	30.9	41.4	10	20.5		7.3	21.1	31.3	41.6	10.2	20.5
11	9.6	14	24.3	10.7		8.7	13.8	23.9	10.1		6.8	21.5	29.1	41.9	7.6	20.4		6.9	20.9	28.9	42.3	8	21.4
12	9.4	19	29.3	10.8		8.5	18.8	29.5	10.7		8.8	20.8	36.6	47.5	10.9	26.7		8.5	21.4	36.5	47.4	11.9	26
13	9.3	17	26.3	9.6		8.8	17.1	26.8	9.7		7.3	21.2	31.9	42.1	10.7	20.9		7.5	21.5	31.9	41.9	10.4	20.4
14	9.3	17	26.4	9.1		9.2	16.9	26.6	9.7		7.3	21.2	31.2	41.9	10	20.7		7.2	21.6	32.9	42.7	9.8	9.8
15	8.8	14	20.3	6.8		8.7	13.8	20.8	7		6.9	20.9	32.1	42.2	11.2	21.3		7.5	21.1	32.3	42.1	11.2	21
16	9.3	17	26.9	10.4		9.2	16.8	27.2	10.4		7.2	21.3	32.1	41.8	10.8	20.5		7.3	20.7	31.5	41.1	10.8	20.4
17	8.8	17	26.9	9.7		9.2	16.9	26.8	9.9		7.3	20.8	32.4	42.1	11.6	21.3		7.2	20.9	32.6	43.1	11.7	22.2
18	9.2	13	20.1	6.9		9.1	13.5	20.8	7.3		7.5	21.6	34.5	46.5	12.9	24.9		7.6	21.1	35.2	44.5	14.1	23.4
19	9.4	14	20.9	7.4		8.6	13.7	21.1	7.4		6.9	20.8	33.5	44.5	12.7	23.7		7.3	21.3	34.2	45.2	12.9	23.9
20	8.9	13	20.8	7.6		9.2	14.1	21.5	7.4		7.2	20.7	34.7	45.3	14	24.6		7.6	20.9	34.9	46.3	14	25.4
21	9.2	17	27.4	10.9		9.7	16.6	28.3	11.7		7.3	21.2	34.8	44.8	13.6	23.6		8.1	21.2	35.3	46.3	11	25.1
22	8.9	17	28.6	11.3		9.4	17.2	28.4	11.2		7.4	21.1	34.8	45.6	13.7	24.5		7.3	21.8	35.8	47.3	14	25.5
23	9.5	18	27.9	10.4		8.6	16.9	27.3	10.4		7.6	20.8	33.9	45.8	13.1	25		7.9	20.7	34.9	46.5	14.2	25.8
24	9.2	19	30.5	12		8.5	18.9	30.8	21.9		7.3	21.2	35.5	46.8	14.3	25.6		7.5	21.3	36.5	46.8	15.2	25.3
25	9.7	17	27.6	11		8.9	17.3	27.8	10.5		7.6	20.9	34.2	46.3	13.3	25.4		7.6	20.5	35.7	46.5	15.2	26
26	8.9	14	20.3	6.6		8.8	13.8	21.6	7.8		7.6	21.2	35.6	45.8	14.4	24.6		7.4	21.2	34.5	45.8	13.3	24.6

	NCM		sex	age	agerange		reflex L	SL	PAIN	P&V	BS	MRI		C3	C4	C5	C6	C6	D1-6	D7-12			
1	priya		2	24	4		5	5	0	0	1	2		0	0	0	0	0	1	0			
2	Rani		2	26	6		7	5	0	0	1	2		0	0	0	0	0	1	0			
3	devaraj		1	28	3		0	5	0	0	1	2		0	0	0	0	0	1	0			
4	balu		1	30	4		2	2	0	0	0	1		1	1	1	0	0	0	0			
5	kanaga		2	38	5		0	5	0	0	1	1		0	0	0	0	0	1	0			
6	kala		2	40	5		7	5	0	0	1	2		0	0	0	0	0	1	0			
7	sivaraj		1	38	4		5	6	0	0	1	2		0	0	0	0	0	0	1			
8	periyannan		1	45	3		0	5	0	0	1	2		0	0	0	0	0	1	0			
9	yasmin		2	24	4		7	5	0	1	1	2		0	0	0	0	0	1	0			
10	susai rani		2	36	2		0	0	1	1	0	0		0	0	0	0	0	0	0			
11	murugan		1	33	5		0	0	1	1	0	0		0	0	0	0	0	0	0			
12	sundaram		1	32	5		0	0	1	1	0	0		0	0	0	0	0	0	0			
13	manikandan		1	34	4		0	0	1	0	0	0		0	0	0	0	0	0	0			
14	nathan		1	42	4		0	0	1	0	0	0		0	0	0	0	0	0	0			
15	marimuthu		1	35	5		0	0	1	0	1	0		0	0	0	0	0	0	0			
16	Gajendran		1	42	4		0	5	0	0	1	2		0	0	0	0	0	1	0			
17	ganesan		1	35	4		7	5	0	0	1	2		0	0	0	0	0	1	0			
18	vidhya		2	29	2		5	6	0	0	1	2		0	0	0	0	0	1	1			
19	munusamy		1	45	1		2	2	0	0	1	1		0	1	1	1	1	0	0			
20	rajendran		1	33	1		5	6	0	0	1	2		0	0	0	0	0	0	1			
21	hari		1	40	4		7	6	0	0	1	2		0	0	0	0	0	0	1			
22	venkatesan		1	34	4		7	5	0	0	1	2		0	0	0	0	0	1	1			
23	egambaram		1	32	5		0	5	0	0	1	2		0	0	0	0	0	1	1			
24	vishwanathan		1	33	4		0	5	0	0	1	2		1	1	1	1	1	0	0			
25	gurusamy		1	38	4		1	1	0	0	1	1		0	0	0	0	0	1	1			
26	lakshmipathy		1	48	5		7	5	0	0	1	2		0	0	0	0	0	1	1			

NON-COMPRESSIVE MYELOPATHY GROUP																							
Sl.No.	Median Right					Median Left					Tibial Right							Tibial Left					
	N9	N13	N20	N13-20		N9	N13	N20	N13-20		N7	N20	N28	P37	N20-N28	N20-P37		N7	N20	N28	P37	N20-N28	N20-P37
1	9.2	14	20.6	7.1		9.1	13.6	20.9	7.3		7.6	21.1	35.4	46.3	14.3	25.2		8.4	21.1	35.5	45.5	14.4	24.4
2	9.5	14	20.8	7		9.5	13.7	21.6	7.9		7.4	21.7	36.4	45.9	14.7	24.2		7.6	21.6	36.4	46.2	14.8	24.6
3	8.9	14	21.6	7.4		8.6	14.7	22.7	8		8.2	20.9	36.8	46.3	15.9	25.4		7.9	20.8	37.2	46.7	16.4	25.9
4	9.2	18	27.9	10.1		8.9	16.8	27.4	10.6		8.5	21.1	36.3	45.3	15.2	24.2		6.9	21.5	37.3	45.8	15.8	24.3
5	8.5	14	21.8	8.1		8.8	13.9	20.9	7		8.8	21.8	35.8	46.9	14	25.1		7.5	20.7	36.1	45.9	15.4	25.2
6	8.7	14	22.1	8.2		8.9	14.3	22.1	7.8		7.3	21.2	35.6	45.8	14.4	24.6		7.4	20.6	37.1	45.3	16.5	24.7
7	8.5	14	21.2	7.7		8.8	13.5	21.4	7.9		7.2	20.9	36.5	46.3	15.6	25.4		7.8	21.1	35.7	46.6	14.6	25.5
8	8.9	14	21.5	7.8		9.3	13.6	21.1	7.5		6.8	20.8	36.4	47.3	15.6	26.5		7.5	21.6	37.3	46.2	15.7	24.6
9	9.7	14	22.1	8.4		9.1	13.2	21.7	8.5		7.5	20.9	37.2	46.3	16.3	25.4		6.9	22.2	37.6	46.9	15.4	24.7
10	9.3	13	21.5	8.3		9.3	13.6	22.4	8.8		7.2	21.8	36.5	45.9	14.7	24.1		7.3	21.1	37.3	45.8	16.2	24.7
11	9.2	14	22.1	8.6		9.4	13.7	21.8	8.1		7.3	22.3	37.5	46.5	15.2	24.2		7.6	22.3	37.9	46.8	14.6	24.5
12	8.5	13	21.8	8.4		9.3	13.5	21.5	8		7.4	22.3	37.8	46.3	15.5	24		7.4	21.6	36.3	46.8	14.7	25.2
13	8.3	14	21.2	7.6		8.8	13.4	21.6	8.2		6.7	21.7	36.7	45.9	15	24.2		6.8	21.5	36.8	46.3	15.3	24.8
14	8.5	13	22.1	7.7		8.3	13.4	22.2	7.8		7.2	21.9	36.3	45.8	14.4	13.9		7.4	22.1	36.8	46.7	14.7	24.6
15	8.9	14	21.9	7.7		8.7	13.6	21.8	8.2		7.5	21.2	35.6	45.4	14.4	24.2		7.3	21.6	36.9	45.7	15.3	24.1
16	8.3	14	21.8	8.1		8.5	13.8	22.8	9		7.3	20.9	35.8	43.4	14.9	22.5		7.5	20.9	36.2	44.3	15.3	23.4
17	9.5	14	21.8	8.2		8.9	13.7	21.8	8.1		7.5	20.7	33.2	44.7	12.5	24		7.8	21.1	35.8	45.3	14.7	24.2
18	8.8	14	22.1	7.5		8.3	13.2	22.3	9.1		7.2	21.6	34.3	44.2	12.7	22.6		7.4	21.4	35.2	44.3	13.8	22.9
19	8.7	14	21.6	7.8		8.2	13.4	21.7	8.3		7.4	20.6	33.6	45.7	13	25.1		7.3	20.6	35.4	45.5	14.8	14.9
20	8.3	14	22.1	8.6		8.6	13.4	20.8	7.4		7.3	20.4	34.6	45.7	14.2	25.3		7.4	20.8	35.7	43.5	14.9	22.7
21	8.4	13	21.5	8.7		8.3	12.6	20.9	8.3		7.5	21.1	33.9	45.2	12.8	24.1		7.1	20.6	35.2	44.7	14.6	24.1
22	9.4	14	22.2	8.4		9.2	13.7	20.9	7.2		7.8	21.1	35.3	44.8	14.2	23.7		7.4	21.1	36.2	45.4	15.1	24.3
23	9.3	13	21.6	8.4		9.1	13.6	20.8	7.2		7.5	20.8	35.7	44.9	14.9	24.1		6.8	20.8	35.8	44.5	15	13.7
24	9.2	14	21.8	8.2		9.3	13.3	20.7	7.4		7.5	20.6	34.9	43.9	14.3	23.3		7.2	20.9	34.2	45.3	13.3	24.4
25	9.1	14	21.6	7.8		9.2	13.2	20.6	7.4		7.8	21.1	35.7	44.7	14.6	23.6		6.8	20.8	35.6	45.3	14.8	24.5
26	8.8	13	22.1	8.5		8.7	13.5	21.2	7.7		6.9	20.8	35.9	45.7	15.1	23.9		7.2	20.9	35.3	46.8	14.4	25.9

						MEDIAN RIGHT					MEDIAN LEFT					TIBIAL RIGHT						
	CONTROL		Sex	age	agerang	N9	N13	N20	N13-20		N9	N13	N20	N13-20		N 7	N20	N28	P37	N20-28	N20-P37	N7
1	Ilayaraja		1	33	2	9.1	13.2	19.8	6.6		9.2	13.1	20	6.9		7.1	23	28.5	39.5	6	17	7
2	perianna		1	38	2	8.8	13.1	18.5	5.4		9.3	12.8	19.5	6.7		6.8	22	27.3	38.2	5.8	16.7	6.7
3	rajan		1	35	2	8.6	12.5	18.7	6.2		8.7	13.3	20.5	7.2		6.9	20	27.2	36.5	7.7	17	6.6
4	vijaya		2	40	3	8.5	13.4	20.2	6.8		8.5	12.6	19.3	6.7		6.5	19	26.5	38.3	7.7	19.5	7.4
5	devi		2	25	1	9.6	13.6	21.6	8		9.4	12.5	21.2	8.7		8.1	21	27.5	39.2	7	18.7	7.3
6	ramu		1	22	1	9.2	13.9	21.4	7.5		8.5	13.2	20.8	7.6		7.3	20	27.3	37.4	6.9	17	8.3
7	venkatesan		1	45	3	8.3	12.3	19.2	6.9		8.2	13.8	20.6	6.8		7.8	21	27.8	39.2	6.4	17.8	7.8
8	daniel		1	23	1	9.5	14.1	21.1	7		9.1	13.9	21.7	7.8		7.5	21	28.5	38.8	7.2	17.5	6.8
9	rani		2	28	1	8.3	13.8	19.2	5.4		9.3	14.2	21.3	7.1		7.2	21	29.2	38.4	8.4	17.6	7.6
10	lakshmi		2	21	1	8.3	12.5	20.5	8		8.6	13.4	20.5	7.1		8.2	22	28.3	38.5	6.3	16.8	8.1
11	poongodi		2	44	3	9.3	12.7	21.1	8.4		9.2	13.6	20.7	7.1		6.8	20	28.6	38.2	8.3	17.9	7.2
12	prakash		1	38	2	9.2	12.9	20.5	7.6		9.3	13.2	20.6	7.4		6.9	21	28.3	37.4	7.7	16.7	7.4
13	sivakumar		1	52	4	9.4	13.5	20.8	7.3		8.9	12.8	21.2	8.4		6.7	22	29.8	38.7	8	16.9	6.8
14	manimaran		1	18	1	8.7	12.6	19.4	6.8		9.3	13.9	21.3	7.4		7.9	22	29.5	39.8	7.2	17.5	7.3
15	ragahavan		1	55	4	9.5	13.8	21.6	7.8		8.8	14.2	21.5	7.3		6.8	23	30.3	36.7	7.8	14.2	7.5
16	manickam		1	43	3	8.6	13.2	21.7	8.5		8.5	13.8	21.5	7.7		7.3	20	28.3	37.9	7.9	17.5	6.8
17	kumaravel		1	38	2	8.6	12.5	20.9	8.4		8.6	12.6	21.2	9.2		6.7	21	28.3	37.3	7.7	16.7	7.2
18	jaya		2	26	1	8.6	13.3	20.8	7.5		9.2	13.5	21.1	7.6		7.3	21	28.9	36.7	8.4	16.2	7.4
19	manjula		2	43	3	8.8	13.6	20.9	7.3		8.7	13.6	20.7	7.1		6.8	21	28.3	37.5	7.8	17	7.2
20	prabhu		1	20	1	9.3	13.6	20.8	7.2		9.2	13.5	20.6	7.1		7.2	21	28.7	37.6	8	16.9	7.3

TIBIAL LEFT				
N20	N28	P37	N20-28	N20-37
22.2	29.3	39	7.1	16.8
19.6	27.5	38.5	7.9	18.9
22.2	28.7	38.6	6.5	16.4
21.8	28.5	39.8	6.7	18
21.5	28.3	36.8	6.8	15.3
21.8	27.3	37.8	5.5	16
21.4	27.5	38.8	6.1	17.4
22.1	28.3	39.2	6.2	17.1
21.7	28.7	38.5	7	16.8
20.2	27.6	37.8	7.4	17.6
20.4	28.3	38.2	7.9	17.8
20.3	28.2	37.5	7.9	17.2
22.2	29.8	38.5	7.6	16.3
20.9	27.7	39.8	6.8	18.9
19.5	28.5	38.3	9	18.8
19.7	27.6	36.8	7.9	17.1
20.5	28.7	38.2	8.2	17.7
19.5	28.3	37.5	8.8	18
19.4	28.9	38.3	9.5	18.9
20.5	28.6	37.6	8.1	17.1

ABBREVIATIONS

1-male, 2- female	BS- Bladder symptoms 1-present 0-absent
age range :(20-30)-1.(30-40)-2(40-50)-3(50-60)-4(60-70	MRI CC(Cord compression)- C3-1, C4-5,D1-6: 6;D7-12:7
reflexL:TrJ-1,BJ-2,SJ-3,TJ-4,Abd(U)-5,Abd(L)-6, BJ+SJ-8	Mrsignal:C3-1;C4-2;C5-3;C6-4;C7-5;D1-6: 6; D7-12:7
SL-sensory level- C4-1;C5-2;C6-D1-6:5, D7-12:6	
Pain sensation:1-present;0-absent	
P&V-position and vibraton-1-present 0-absent	
MRI- 1-CERVICAL ,2-THORACIC	
CM - Compressive Myelopathy	
NCM - Non Compressive Myelopathy	